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Editorial

Early treatment of unstable angina

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There is considerable controversy about what is the most appropriate treatment for patients with unstable angina. Some workers suggest that a conservative approach is all that is necessary[1]; others support the early use of investigative techniques, angioplasty, and operation.[2] Much of this conflict stems from difficulty with the exact definition of unstable angina. Ambulatory monitoring in patients with stable angina has shown that ST segment changes with and without chest pain frequently occur when the patients are apparently at rest; approximately 15% of patients with stable angina and severe coronary artery disease have nocturnal evidence of myocardial ischaemia.[3] Such patients, in whom progression to an acute myocardial infarction is rare, would not normally be regarded as having unstable angina or pre-infarction. If they were to attend a casualty department, however, this would invariably be the diagnosis. At the other end of the spectrum are those patients in the early stages of acute myocardial infarction in whom there is a stuttering onset in the hours before the development of myocardial cell necrosis. The benefits to be expected from treatment and the most appropriate technique of management will be quite different in these two disparate groups of patients despite their common diagnosis of unstable angina. In patients with severe angina, treatment with nitrates, β blockers, and calcium antagonists would theoretically be the best approach; in contrast, in the stuttering early stages of myocardial infarction anything short of rapid reversal of the thrombotic process by aspirin, streptokinase, or tissue-type plasminogen activator is likely to be of little avail. Some patients will also require restoration of coronary blood flow by percutaneous transluminal angioplasty or coronary bypass graft surgery.

Because of the known pathophysiological mechanisms in stable angina patients with this condition can be reliably identified on the basis of their clinical history, exercise testing, ambulatory monitoring, and coronary anatomy. The effects of different drugs in stable angina may then be objectively evaluated in double blind control studies of homogeneous groups of patients.[4] Such results have useful clinical implications which have been borne out by subsequent clinical practice. In contrast, studies in patients with unstable angina are very likely to involve a heterogeneous group with different pathophysiological mechanisms. In this issue (1986;**56**:400–13), the Holland Interuniversity Nifedipine/metoprolol Trial research group report the results of their study.[5] They have examined the effects of placebo, nifedipine, metoprolol, and a combination of the two in 338 patients with unstable angina not pre-treated with a β blocker and a further 177 patients pre-treated with a β blocker. The study was discontinued because nifedipine was suspected of increasing the frequency of myocardial infarction. These results, however, must be interpreted with caution. Although the myth that β blockers are contraindicated in unstable angina because of the theoretical risk of coronary spasm can now be laid to rest, the case that nifedipine is detrimental is far from proved. The number of patients in this study in whom acute myocardial infarction developed within a few hours of admission suggests that a substantial proportion of the patients had stuttering early infarction. In this group aspirin, streptokinase, and angioplasty or bypass grafting might have been more appropriate.

Fortunately, the findings of this and early studies provide important guidelines for clinical practice. As clinicians we are not really concerned with whether nifedipine is more effective than a β blocker or vice versa because almost all patients with unstable angina are vigorously treated with intravenous nitrates, oral β blockers, an oral calcium antagonist,

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and aspirin in combination. In patients who do not respond very rapidly to this approach angioplasty or surgery are the treatments of choice.

References

1 Mulcahy R, Daly G, Graham I, *et al*. Unstable angina: natural history and determinants of prognosis. *Am J Cardiol* 1981;**48**:525–8.

2 De Feyter PJ, Serruys PW, Van der Brand M, *et al*. Emergency coronary angioplasty in unstable angina. *N Engl J Med* 1985;**313**:342–6.

3 Quyyumi AA, Mockus L, Wright C, Fox KM. Morphology of ambulatory ST segment changes in patients with varying severity of coronary artery disease: investigations of the frequency of nocturnal ischaemia and coronary spasm. *Br Heart J* 1985;**53**:186–93.

4 Lynch P, Dargie HJ, Krikler S, Krikler D. Objective assessment of antianginal therapy: a double blind comparison of nifedipine, propranolol and their combination. *Br Med J* 1980;**281**:184–7.

5 Holland Interuniversity Nifedipine/metoprolol Trial (HINT) research group. Early treatment of unstable angina in the coronary care unit: a randomised, double blind, placebo controlled comparison of recurrent ischaemia in patients treated with nifedipine or metoprolol or both. *Br Heart J* 1986; **56**:400–13.